

abdominal pain, alternating states of consciousness, copious mucous secretions, palpitations, photophobia, muscle twitching, miosis, clammy distal extremities, pulmonary rales and wheezes and hypoactive deep tendon reflexes. During the first 36 hours after admission to hospital 5 percent sugar by Clinitest® and Labstix® (glucose oxidase), and large amounts of acetone in the urine were noted. Results of additional laboratory studies showed leukocytosis, hypokalemia, mild glycosemia and normal serum insulin.

Von Oettingen outlined the symptoms and signs of acute caffeine poisoning as vomiting, epigastric pain, vertigo, extreme miosis, photophobia, flickering before the eyes, diplopia, amblyopia, visual field restriction, confusion and restlessness, headache, insomnia, tremor, delirium, hallucination, tachycardia, hypotension, cold and clammy skin, labored breathing, and diuresis with possible glycosuria and acetonuria. Boyd and associates reported polydipsia, polyuria, and glycosuria in experimental rats after high dose chronic caffeine administration; however, no mention of acetonuria was made. Bronchopneumonia and pulmonary edema were named as the terminal events.

The toxic dose of caffeine in humans is suggested to be between 2 and 20 mg per kg of body weight, while the lethal dose is felt to be between 140 and 200 mg per kg of body weight. Findings in animal studies indicate that an age related susceptibility factor exists with sensitivity to toxic doses increasing with age. The physiologic effects of acute caffeine overdosage can for the most part be explained; however, glycosuria and acetonuria without glucosemia or acetonemia cannot. Boyd reported the histologic finding of extensive kidney inflammation with the brush border of cells lining the renal proximal convoluted tubules appearing necrotic. Red blood cells filled the loops of Henle and cells of the distal convoluted tubules were swollen to seal off the union. This suggests generalized renal damage with lowering of the threshold perhaps to a number of normal plasma constituents; the mildly elevated blood glucose might be explained on the basis of an adrenal stress factor.

We feel that because of the increased use of caffeine containing insomnics (especially since a palatable form exists) physicians need to be alerted to the effects of caffeine in acute overdosage. One must be alert as to the similar clinical features in patients with diabetic ketoacidosis. Supportive measures as used in other forms of

poisoning, such as control of seizures, respiratory arrest, treatment of shock and the use of cardiac depressant drugs may be indicated.

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Meningococcal Prophylaxis

IT HAS LONG BEEN recognized that family members of patients with meningococcal meningitis have an increased risk of contracting the disease. Results of studies of meningococcal carriage rates in contacts have shown an increased likelihood of acquisition of the organism in close family members. The carriage rates drop, however, when compared with schoolroom contacts. For this reason, chemoprophylaxis would seem to be indicated only in close contacts and not for casual acquaintances.

In view of the increasing resistance to the sulfa drugs which the organism has shown since the mid-1960's, these drugs are no longer recommended for chemoprophylaxis unless it is known in advance that the organism is from a sensitive strain. Although penicillin can be used to treat an active case, it has not proved useful in clearing the carrier state.

Recently two antibiotics, minocycline and rifampin, have been used with good results in treating carriers. Rifampin is currently being recommended in a dosage of 600 mg twice daily for two days in adults. The pediatric dosage is 10 mg per kg of body weight twice daily for two days in children between the ages of 1 and 12 years with half that dose in infants between the ages of 3 months and 1 year. Minocycline is no longer recommended due to the high incidence of vestibular side effects, including dizziness, nausea and vomiting.

Monovalent vaccines effective against Types A and C meningococci have recently been licensed

for use in epidemic control. These have been used with success in certain military installations as well as in the recent epidemic in Brazil. Since measurable antibodies do not appear until two or three weeks after inoculation, these vaccines would appear to be of little value in the treatment of close contacts of a patient with an active case.

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